Can J Physiol Pharmacol. 2017 Aug;95(8):888-893. doi: 10.1139/cjpp-2016-0693. Epub 2017 Mar 28.

Molecular hydrogen potentiates beneficial anti-infarct effect of hypoxic postconditioning in isolated rat hearts: a novel cardioprotective intervention

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PMID: 28350967 DOI: 10.1139/cjpp-2016-0693

Abstract

Generation of free radicals through incomplete reduction of oxygen during ischemia-reperfusion (I/R) is well described. On the other hand, molecular hydrogen (H_2) reduces oxidative stress due to its ability to react with strong oxidants and easily penetrate cells by diffusion, without disturbing metabolic redox reactions. This study was designed to explore cardioprotective potential of hypoxic postconditioning (HpostC) against I/R (30 min global I - 120 min R) in isolated rat hearts using oxygen-free Krebs-Henseleit buffer (KHB). Furthermore, the possibility to potentiate the effect of HpostC by H_2 using oxygen-free KHB saturated with H_2 (H_2 + HpostC) was tested. HPostC was induced by 4 cycles of 1-minute perfusion with oxygen-free KHB intercepted by 1-minute perfusion with normal KHB, at the onset of reperfusion. H_2 + HPostC was applied in a similar manner using H_2-enriched oxygen-free KHB. Cardioprotective effects were evaluated on the basis of infarct size (IS, in % of area at risk, AR) reduction, post-I/R recovery of heart function, and occurrence of reperfusion arrhythmias. HPostC significantly reduced IS/AR compared with non-conditioned controls. H_2 present in KHB during HPostC further decreased IS/AR compared with the effect of HPostC, attenuated severe arrhythmias, and significantly restored heart function (vs. controls). Cardioprotection by HpostC can be augmented by molecular hydrogen infusion.

Keywords: antioxidants; antioxydants; hydrogène moléculaire; hypoxic postconditioning; ischemia–reperfusion injury; ischemic postconditioning; lésion d'ischémie–reperfusion; molecular hydrogen; postconditionnement hypoxique; postconditionnement ischémique.

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